AMENDMENT TO THE CLAIMS

This listing of claims will replace all prior versions, and listings, of claims in the applications:

Listing of Claims:

1. (original) A method of prophylaxis therapy for myocardial infarction (MI) comprising:

selecting a human subject susceptible to MI;

administering to the subject a composition comprising a therapeutically effective amount of an MI therapeutic agent that inhibits leukotriene synthesis *in vivo*, wherein the MI therapeutic agent inhibits leukotriene synthesis by inhibiting the activity of at least one protein selected from 5-Lipoxygenase activating protein (FLAP) and 5-lipoxygenase (5-LO), and

monitoring myeloperoxidase (MPO) level in the human subject before and during the prophylaxis treatment, wherein the MI therapeutic agent is administered in an amount effective to reduce the MPO level in a subject.

2. (original) A method according to claim 1, further comprising monitoring at least one additional inflammatory marker in the human subject before and during the prophylaxis therapy.

3. (original) A method according to claim 2, wherein the additional inflammatory marker is C-reactive protein.

4. (original) A method according to claim 1, 2 or 3, wherein the monitoring further comprises monitoring a leukotriene level in serum, plasma, or urine from the human subject before and during the prophylaxis treatment, wherein MI therapeutic agent is administered in an amount effective to reduce the leukotriene level in a subject.

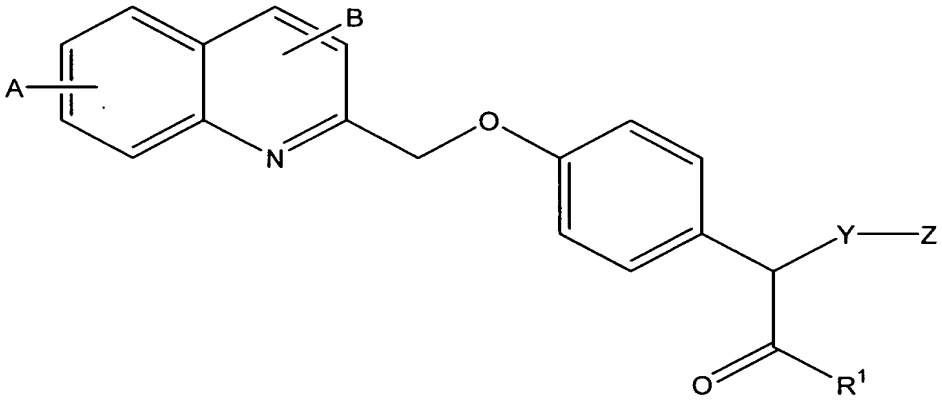
5. (original) A method according to claim 1, 2 or 3, wherein the monitoring comprises measuring leukotriene production *ex vivo* in a blood sample from the human subject.

6. (original) A method according to claim 5, wherein the blood sample is stimulated with a calcium ionophore prior to measuring leukotriene production.

7. (original) A method according to any one of claims 1-6, wherein the MI therapeutic agent inhibits FLAP activity, and the composition is administered in an amount effective to inhibit FLAP polypeptide activity in the human subject.

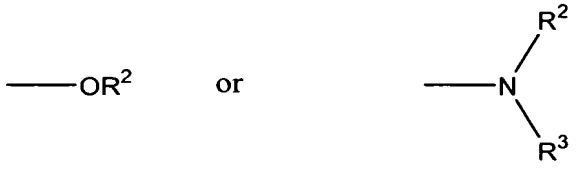
8. (original) A method according to claim 7, wherein the composition further comprises a physiologically acceptable carrier or excipient.

9. (original) The method of claim 7 or 8, wherein the MI therapeutic agent comprises a compound represented by the formula:

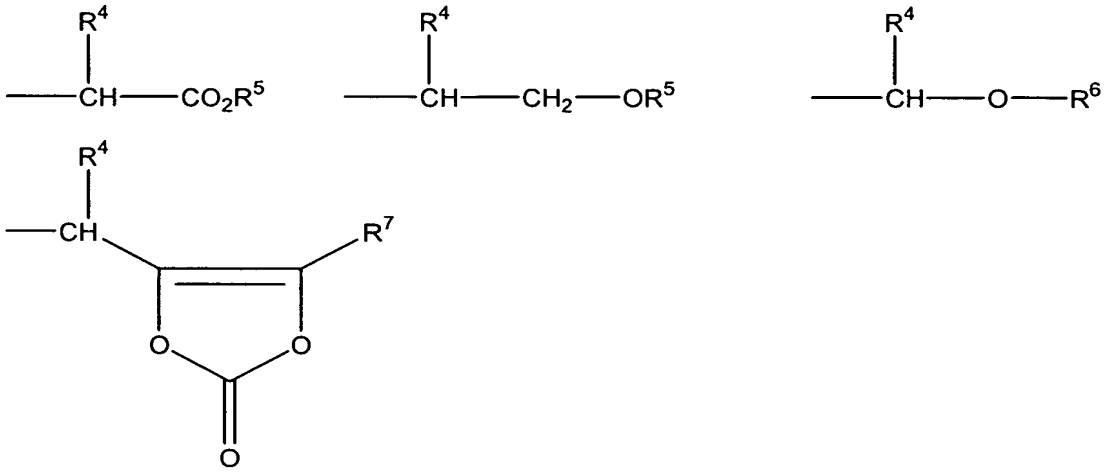


or pharmaceutically acceptable salt thereof ,

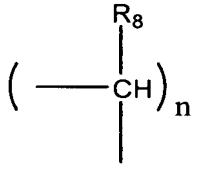
wherein R^1 represents a group of the formula:



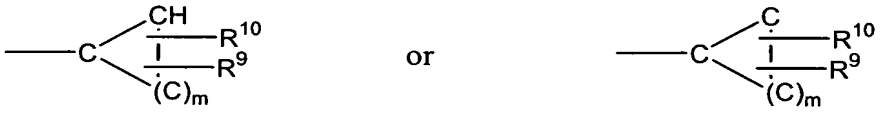
R^2 and R^3 are identical or different and represent hydrogen, lower alkyl, phenyl, benzyl or a group of the formula:



R^4 represents hydrogen, lower alkyl, phenyl or benzyl, which can optionally be substituted by hydroxyl, carboxyl, lower alkoxycarbonyl, lower alkylthio, heteroaryl or carbamoyl, R^5 represents hydrogen, lower alkyl, phenyl or benzyl, R^6 represents a group of the formula $-COR^5$ or $-CO^2 R^5$, R^7 represents hydrogen, lower alkyl or phenyl, Y represents a group of the formula:



wherein R^8 represents hydrogen, lower alkyl or phenyl and n denotes a number of 0 to 5, Z represents norbornyl, or represents a group of the formula:



wherein R^9 and R^{10} are identical or different and denote hydrogen, lower alkyl or phenyl, or R^9 and R^{10} can together form a saturated carbocyclic ring having up to 6 carbon atoms and m denotes a number from 1 to 6, and A and B are identical or different and denote hydrogen, lower alkyl or halogen, or a pharmaceutically acceptable salt thereof.

10. (original) The method according to claim according to claim 7 or 8, wherein the MI therapeutic agent comprises a compound selected from the group consisting of: 2-[4-(quinolin-2-yl-methoxy)phenyl]-2-cyclopentylacetic acid, 2-[4-(quinolin-2-yl-methoxy)phenyl]-2-cyclohexylacetic acid, and 2-[4-(quinolin-2-yl-methoxy)phenyl]-2-cycloheptylacetic acid, (+)-enantiomer of 2-[4-(quinolin-2-yl-methoxy)phenyl]-2-cyclopentylacetic acid, (-)-enantiomer of 2-[4-(quinolin-2-yl-methoxy)phenyl]-2-cyclopentylacetic acid, and pharmaceutically acceptable salts thereof.

11. (original) The composition according to claim 7 or 8, wherein the MI therapeutic agent comprises BAY-X-1005 or a physiologically acceptable salt, formulation, or pro-drug thereof.

12. (original) A method according to any one of claims 1-11, wherein the MI therapeutic agent is administered in an amount effective to reduce the leukotriene level in the subject lower than a median level of leukotrienes in human subjects.

13. (original) A method according to any one of claims 1-12, wherein the selecting step comprises selecting a susceptible subject from an elevated measurement of at least one inflammatory marker selected from the group consisting of C-reactive protein (CRP), serum amyloid A, fibrinogen, interleukin-6, tissue necrosis factor-alpha (TNF-alpha), soluble vascular cell adhesion molecules (sVCAM), soluble intervascular adhesion molecules (sICAM), E-selectin, matrix metalloprotease type-1, matrix metalloprotease type-2, matrix metalloprotease type-3, matrix metalloprotease type-9, myeloperoxidase (MPO), and N-tyrosine.

14. (original) A method according to claim 13 or 14, wherein the MI therapeutic agent is administered in an amount effective to reduce the elevated serum level of at least one inflammatory marker.

15. (original) A method according to claim 13, wherein the measurement is a measurement from serum from the subject.

16. (original) A method according to any one of claims 13-15, wherein the measurement is a measurement of myeloperoxidase.

17. (original) A method according to any one of claims 1-12, wherein the selecting step comprises selecting a susceptible subject from at least one family or medical history risk factor selected from the group consisting of past or current smoker; diabetes; hypertension; serum total cholesterol > 200mg/dL; elevated serum LDL cholesterol; low serum HDL cholesterol; elevated C-reactive protein (CRP); elevated serum amyloid A; hypercholesterolemia; elevated triglycerides; elevated lp(a); obesity; acute coronary syndrome (ACS); angina; atherosclerosis; ankle/brachial index less than 0.9; transient ischemic attack; transient monocular blindness; asymptomatic carotid stenosis; claudication; limb ischemia leading to gangrene, ulceration or amputation; surgery or stent to restore coronary artery blood flow and angioplasty.

18. (original) A method according to any one of claims 1-12, wherein the selecting comprises selecting a susceptible subject from an elevated measurement of a leukotriene or leukotriene metabolite from the subject.

19. (original) A method according to claim 18, wherein the selecting step comprises selecting a susceptible subject from an elevated measurement of a leukotriene in a biological sample from the subject, said leukotriene selected from the group consisting of LTC4, LTD4, LTB4, and LTE4.

20. (original) A method according to claim 18 or 19, wherein the selecting step comprises selecting a susceptible subject from an elevated measurement of leukotriene E4 (LTE4) in serum, plasma, or urine from the subject.

21. (original) A method according to claim 18 or 19, wherein the selecting step comprises selecting a susceptible subject from an elevated leukotriene measurement in blood from the subject.

22. (original) A method according to claim 21, wherein leukotriene production in the blood is stimulated with a calcium ionophore prior to measuring the leukotriene.

23. (original) A method according to any one of claims 1-12, where the selecting comprises determining a FLAP genotype or haplotype of a human subject, and selecting for treatment a human subject with a FLAP genotype or haplotype that correlates with an increased risk of myocardial infarction.

24. (original) A method according to any one of claims 1-12, wherein the selecting comprises analyzing nucleic acid of a human subject for the presence or absence of at least one 5-lipoxygenase activating protein (FLAP) polymorphism that correlates with a susceptibility to myocardial infarction.

25. (original) A method according to claim 23 or 24, wherein the selecting step further comprises analyzing serum CRP or MPO, and selecting a subject with the presence of at least one such FLAP genotype, haplotype or polymorphism and with the presence of elevated serum CRP or MPO.

26. (original) A method according to any one of claims 23, wherein the selecting comprises selecting a human subject having a FLAP (SEQ ID NO: 1) haplotype comprising:

SG13S377 (SEQ ID NO: 1, position 169965), allele A

SG13S114 (SEQ ID NO: 1, position 178096), allele A;

SG13S41 (SEQ ID NO: 1, position 202045), allele A; and

SG13S35 (SEQ ID NO: 1, position 206117), allele G.

27. (original) A method according to claim 26, wherein the FLAP (SEQ ID NO: 1) polymorphism further comprises SG13S375 (SEQ ID NO: 1, position 164874), allele T.

28. (original) A method according to claim 26 or 27, wherein the presence in said subject of a haplotype further comprised of marker SG13S106 (SNP DG00AAHII) (SEQ ID NO: 1, position 176579), allele G, identifies the subject as having a susceptibility to MI.

29. (original) A method according to claim 26 or 27, wherein the presence in said subject of a haplotype further comprised of marker SG13S106 (SNP DG00AAHII) (SEQ ID NO: 1, position 176579), allele G; SG13S30 (SEQ ID NO: 1, position 193840), allele G; and SG13S42 (SEQ ID NO: 1, position 203877), allele A, identifies the subject as having a susceptibility to MI.

30. (original) A method of prophylaxis therapy for myocardial infarction (MI) comprising:

selecting a human subject having a FLAP (SEQ ID NO: 1) haplotype comprising: SG13S375 (SEQ ID NO: 1, position 164874), allele T;

administering to the subject a composition comprising a therapeutically effective amount of an MI therapeutic agent that inhibits leukotriene synthesis *in vivo*, wherein the MI therapeutic agent inhibits leukotriene synthesis, and

monitoring myeloperoxidase (MPO) level in the human subject before and during the prophylaxis treatment, wherein the MI therapeutic agent is administered in an amount effective to reduce the MPO level in a subject.

31. (original) A method according to claim 30, wherein the selecting comprises selecting a human subject having a FLAP (SEQ ID NO: 1) haplotype further comprising:

SG13S25 (SEQ ID NO: 1, position 165553), allele G;

SG13S32 (SEQ ID NO: 1, position 176579), allele A; and

SG13S106 (SEQ ID NO: 1, position 198547), allele G or A.

32. (original) A method according to any one of claim 30 or 31, wherein the presence in said subject of a haplotype further comprised of markers:

SG13S25 (SEQ ID NO: 1, position 165553), allele G;

SG13S99 (DG00AAFIU), allele T (SEQ ID NO: 1, position 138551);

SG13S377 (DG00AAJFF) (SEQ ID NO: 1, position 169965), allele G;

SG13S106 [SNP DG00AAHII] (SEQ ID NO: 1, position 176579), allele G;

SG13S32 (SEQ ID NO: 1, position 198547), allele A; and

SG13S35 (SEQ ID NO: 1, position 206117), allele G,

identifies the subject as having a susceptibility to MI.

33. (original) A method according to claim 23, wherein the presence in said subject of a haplotype comprised of markers:

SG13S377 (SEQ ID NO: 1, position 169965), allele A;

SG13S114 (SEQ ID NO: 1, position 178096), allele A;

SG13S41 (SEQ ID NO: 1, position 202045), allele A; and

SG13S35 (SEQ ID NO: 1, position 206117), allele G,

identifies the subject as having a susceptibility to MI.

34. (original) A method according to claim 23, wherein the presence in said subject of a haplotype comprised of markers:

SG13S375 (SEQ ID NO: 1, position 164874), allele T

SG13S25 (SEQ ID NO: 1, position 165553), allele G;

SG13S32 (SEQ ID NO: 1, position 176579), allele A; and

SG13S106 (SEQ ID NO: 1, position 198547), allele G or A,

identifies the subject as having a susceptibility to MI.

35. (original) A method according to any one of claims 31-34, wherein the presence in said subject of a haplotype further comprised of marker SG13S375(SNP DG00AAJFC) (SEQ ID NO: 1, position 164874), allele T; and SG13S25 (SEQ ID NO: 1, position 165553), allele G, identifies the subject as having a susceptibility to MI.

36. (original) A method according to any one of claims 31-34, wherein the presence in said subject of a haplotype further comprised of marker SG13S375(SNP DG00AAJFC) (SEQ ID NO: 1, position 164874), allele T; and SG13S25 (SEQ ID NO: 1, position 165553), allele G, and SG13S32 (SEQ ID NO: 1, position 198547) identifies the subject as having a susceptibility to MI.

37. (currently amended) A method of prophylaxis therapy for myocardial infarction (MI) comprising:

analyzing nucleic acid of a human subject for the presence and absence of a FLAP haplotype, wherein the haplotype is comprised of markers:

~~SG13S37~~ SG13S375 (SEQ ID NO: 1, position 164874), allele T;

SG13S25 (SEQ ID NO: 1, position 165553), allele G;

SG13S32 (SEQ ID NO: 1, position 176579), allele A; and

SG13S106 (SEQ ID NO: 1, position 198547206117), allele G or A,

selecting for treatment a human subject having nucleic acid with the presence of the FLAP haplotype,

administering to the subject a composition comprising a therapeutically effective amount of an MI therapeutic agent that inhibits leukotriene synthesis *in vivo*, wherein the MI therapeutic agent inhibits leukotriene synthesis.

38. (original) A method according to claim 37, comprising monitoring at least one inflammatory marker in the human subject before and during the prophylaxis therapy.

39. (original) A method according to claim 38 wherein the inflammatory marker is C-reactive protein or myeloperoxidase (MPO).

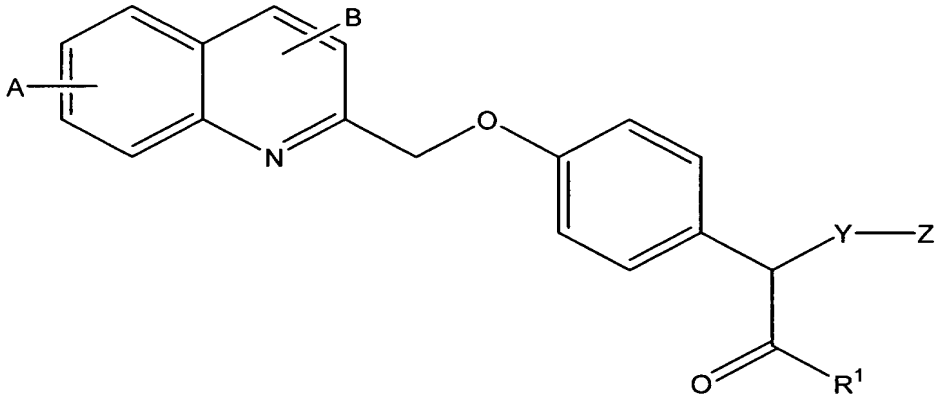
40. (original) A method according to claim 38 or 39, wherein the monitoring comprises monitoring a leukotriene level in serum, plasma, or urine from the human subject before and during the prophylaxis treatment, wherein MI therapeutic agent is administered in an amount effective to reduce the leukotriene level in a subject.

41. (original) A method according to claim 40, wherein the monitoring comprises measuring leukotriene production *ex vivo* in a blood sample from the human subject.

42 (original) A method according to claim 41, wherein the blood sample is stimulated with a calcium ionophore prior to measuring leukotriene production.

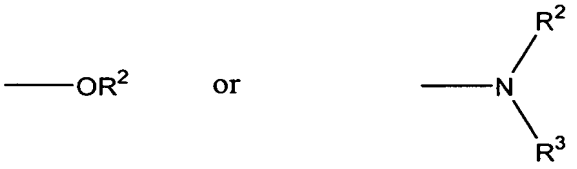
43. (original) A method according to any one of claims 37-42, wherein the MI therapeutic agent inhibits FLAP activity or 5-LO activity.

44. (original) The method of claim 43, wherein the MI therapeutic agent comprises a compound represented by the formula:

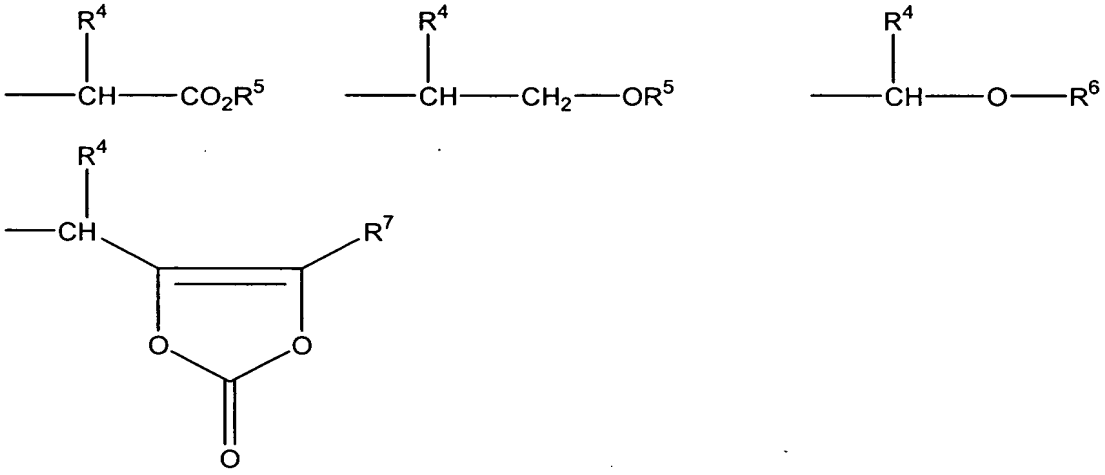


or pharmaceutically acceptable salt thereof ,

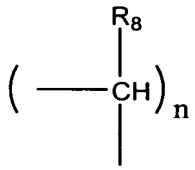
wherein R^1 represents a group of the formula:



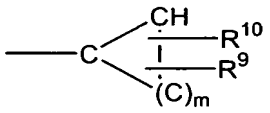
R^2 and R^3 are identical or different and represent hydrogen, lower alkyl, phenyl, benzyl or a group of the formula:



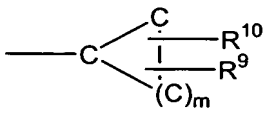
R^4 represents hydrogen, lower alkyl, phenyl or benzyl, which can optionally be substituted by hydroxyl, carboxyl, lower alkoxycarbonyl, lower alkylthio, heteroaryl or carbamoyl, R^5 represents hydrogen, lower alkyl, phenyl or benzyl, R^6 represents a group of the formula -COR^5 or -CO^2 R^5, R^7 represents hydrogen, lower alkyl or phenyl, Y represents a group of the formula:



wherein R^8 represents hydrogen, lower alkyl or phenyl and n denotes a number of 0 to 5, Z represents norbornyl, or represents a group of the formula:



or



wherein R^9 and R^{10} are identical or different and denote hydrogen, lower alkyl or phenyl, or R^9 and R^{10} can together form a saturated carbocyclic ring having up to 6 carbon atoms and m denotes a number from 1 to 6, and A and B are identical or different and denote hydrogen, lower alkyl or halogen, or a pharmaceutically acceptable salt thereof.

45. (original) A method according to any one of claims 37-44, wherein the MI therapeutic agent is administered in an amount effective to reduce the leukotriene level in the subject lower than a median level of leukotrienes in human subjects.

46. (original) A method of prophylaxis for myocardial infarction (MI) comprising:

administering to a subject in need of prophylaxis for myocardial infraction a composition comprising a therapeutically effective amount of an MI therapeutic agent that inhibits leukotriene synthesis *in vivo*, and

monitoring myeloperoxidase (MPO) level in the human subject before and during the prophylaxis treatment, wherein the MI therapeutic agent is administered in an amount effective to reduce the MPO level in a subject.

47. (original) A method of screening a human subject for risk of developing myocardial infarction, comprising:

contacting a blood sample from the human subject with a calcium ionophore to stimulate production of a leukotriene; and

measuring production of a leukotriene in the blood sample after the contacting step, wherein elevated leukotriene production compared to a control correlates with increased risk of developing myocardial infarction (MI).

48. (original) A method according to claim 47, wherein the control comprises a leukotriene measurement from calcium ionophore treated blood from humans of the same sex as the human subject.

49. (original) A method according to claim 47, wherein the control comprises a leukotriene measurement from calcium ionophore treated blood from a human that is age matched to the human subject.

50. (original) A method according to claim 48 or 49, wherein the blood sample comprises isolated blood neutrophils.

51. (original) A method according to any one of claims 47-50, wherein the leukotriene is at least one member selected from LTE4, LTD4, and LTB4.

52. (original) A method according to any one of claims 47-51, further comprising prophylactically administering an MI therapeutic agent to a human subject identified as having increased risk of developing MI, wherein the MI therapeutic agent inhibits leukotriene synthesis by inhibiting the activity of at least one protein selected from 5-Lipoxygenase activating protein (FLAP) and 5-lipoxygenase (5-LO).

53. (original) A method of decreasing risk of a subsequent myocardial infarction in an individual who has had at least one myocardial infarction, comprising

administering a therapeutically effective amount of an MI therapeutic agent to the individual, wherein the MI therapeutic agent inhibits leukotriene synthesis by inhibiting the activity of at least one protein selected from 5-Lipoxygenase activating protein (FLAP) and 5-lipoxygenase (5-LO) and

monitoring myeloperoxidase (MPO) in the individual before and during the administration of the therapeutic agent, wherein the MI therapeutic agent is administered in an amount effective to reduce the MPO level in a subject.

54. (original) A method of screening a human subject for susceptibility for MI comprising

analyzing nucleic acid of a human subject for the presence and absence of the FLAP haplotype comprised of markers:

SG13S377 (SEQ ID NO: 1, position 169965), allele A;

SG13S114 (SEQ ID NO: 1, position 178096), allele A;

SG13S41 (SEQ ID NO: 1, position 202045), allele A; and

SG13S35 (SEQ ID NO: 1, position 206117), allele G,

identifying the subject as having a susceptibility to MI, wherein the presence of the FLAP haplotype correlates with an increased risk of myocardial infarction.

55. (original) A composition comprising a leukotriene synthesis inhibitor and a statin.

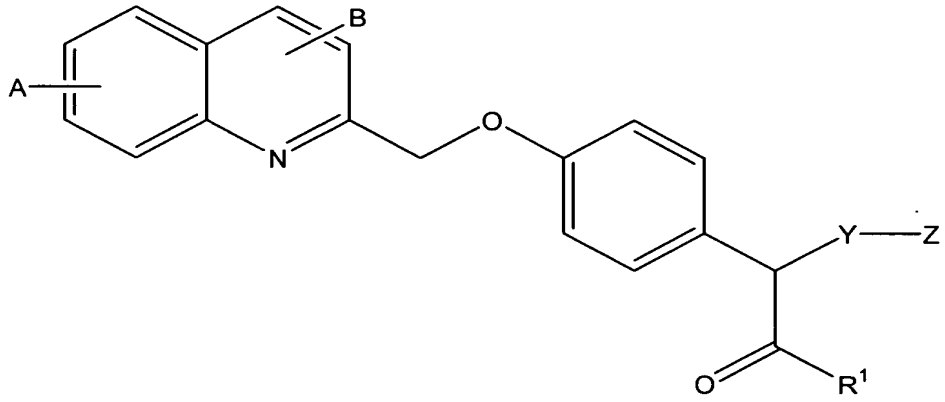
56. (original) A composition according to claim 55, further comprising a pharmaceutically acceptable carrier.

57. (original) The composition according to claim 55 or 56, wherein the leukotriene synthesis inhibitor is an agent that inhibits activity of a leukotriene synthesis pathway protein selected from the group consisting of 5-lipoxygenase, 5-lipoxygenase activating protein (FLAP), leutokriene C4 synthase, leukriene A4 hydolase, arachidonate 4-lipoxygenase, leukotriene B4 12-hydroxydehydrogenase; leukotriene A4 receptor, leukotriene B4 receptor, leukotriene C4 receptor, leukotriene D4 receptor, leukotriene E4 receptor, leukotriene B4 receptor 1, leukotriene B4 receptor 2, cysteinyl leukotriene receptor 1, and cysteinyl leukotriene receptor 2.

58. (original) The composition according to any one of claims 55-57, wherein the leukotriene synthesis inhibitor is selected from the group consisting of 1-((4-chlorophenyl)methyl)-3-((1,1-dimethylethyl)thio)-alpha,alpha-dimethyl-5-(2-quinolinylmethoxy)- 1H-Indole-2-propanoic acid, (R)-(+)-alpha-cyclopentyl-4-(2-quinolinylmethoxy)-Benzeneacetic acid, 3-(3-(1,1-dimethylethylthio-5-(quinoline-2-ylmethoxy)-1-(4-chloromethylphenyl)indole-2-yl)-2,2-dimethylpropionaldehyde oxime-0-2-acetic acid, zileuton, atreleuton, 6-((3-fluoro-5-(tetrahydro-4-methoxy-2H-pyran-4yl)phenoxy)methyl)-1-methyl-2(1H)-quinlolinone, 1-((4-chlorophenyl)methyl)-3-((1,1dimethylethyl)thio)-alpha,alpha-dimethyl-5-(2-quinolinylmethoxy)-1H-Indole-2-propanoic acid and 4-(3-(4-(2-Methyl-imidazol-1-yl)-phenylsulfanyl)-phenyl)-tetrahydro-pyran-4-carboxylic acid amide.

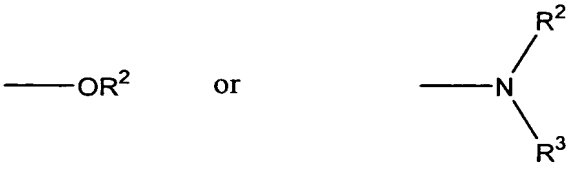
59. (original) The composition according to any one of claims 55-57, wherein the leukotriene synthesis inhibitor is a FLAP inhibitor.

60. (original) The composition according to claim 59, wherein the FLAP inhibitor comprises a compound represented by the formula:

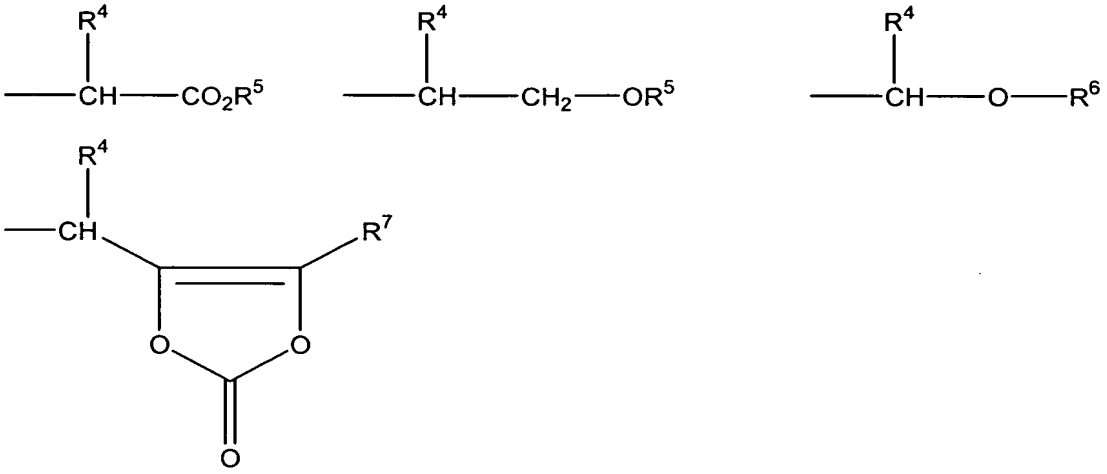


or pharmaceutically acceptable salt thereof ,

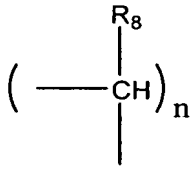
wherein R^1 represents a group of the formula:



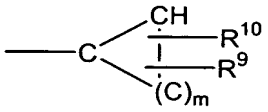
R^2 and R^3 are identical or different and represent hydrogen, lower alkyl, phenyl, benzyl or a group of the formula:



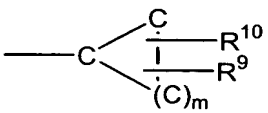
R^4 represents hydrogen, lower alkyl, phenyl or benzyl, which can optionally be substituted by hydroxyl, carboxyl, lower alkoxycarbonyl, lower alkylthio, heteroaryl or carbamoyl, R^5 represents hydrogen, lower alkyl, phenyl or benzyl, R^6 represents a group of the formula -COR^5 or -CO^2 R^5, R^7 represents hydrogen, lower alkyl or phenyl, Y represents a group of the formula:



wherein R^8 represents hydrogen, lower alkyl or phenyl and n denotes a number of 0 to 5, Z represents norbornyl, or represents a group of the formula:



or



wherein R^9 and R^{10} are identical or different and denote hydrogen, lower alkyl or phenyl, or R^9 and R^{10} can together form a saturated carbocyclic ring having up to 6 carbon atoms and m denotes a number from 1 to 6, and A and B are identical or different and denote hydrogen, lower alkyl or halogen, or a pharmaceutically acceptable salt thereof.

61. (original) The composition according to claim 59, wherein the FLAP inhibitor comprises a compound selected from the group consisting of: 2-[4-(quinolin-2-yl-methoxy)phenyl]-2-cyclopentylacetic acid, 2-[4-(quinolin-2-yl-methoxy)phenyl]-2-cyclohexylacetic acid, and 2-[4-(quinolin-2-yl-methoxy)phenyl]-2-cycloheptylacetic acid, (+)-enantiomer of 2-[4-(quinolin-2-yl-methoxy)phenyl]-2-cyclopentylacetic acid, (-)-enantiomer of 2-[4-(quinolin-2-yl-methoxy)phenyl]-2-cyclopentylacetic acid, and pharmaceutically acceptable salts thereof.

62. (original) The composition according to claim 59, wherein the FLAP inhibitor comprises BAY-X-1005 or a physiologically acceptable salt, formulation, or pro-drug thereof.

63. (original) The composition according to claim 59, wherein the leukotriene synthesis inhibitor is (R)-(+)-alpha-cyclopentyl-4-(2-quinolinylmethoxy)-Benzeneacetic acid.

64. (original) The composition according to any one of claims 55-63, wherein the statin is selected from the group consisting of rovuvastatin, fluvastatin, atorvastatin, lovastatin, simvastatin, pravastatin or pitavastatin.

65. (original) The composition according to any one of claims 55-64, wherein the leukotriene synthesis inhibitor is included in the composition in an amount effective to reduce serum C-reactive protein (CRP) in a human subject.

66. (original) The composition according to any one of claims 55-65 wherein the statin is included in the composition in an amount effective to reduce serum low density lipoprotein cholesterol (LDL) and reduce serum CRP in a human subject.

67. (original) The composition according to any one of claims 55-65, wherein the leukotriene inhibitor and the statin are included in the composition in amounts effective to synergistically reduce serum C-reactive protein in a human subject.

68. (original) The composition of any one of claims 55-67 that comprises a unit dose for administration to a human subject.

69. (original) The composition of any one of claims 55-68 that is a pill or capsule.

70. (original) The composition according to claim 68 or 69, wherein 50 to 750 milligrams of the FLAP inhibitor is present in the unit dose.

71. (original) The composition according to claim 68 or 69, wherein 250 to 375 milligrams of the FLAP inhibitor is present in the unit dose.

72. (original) The composition according to claim 68 or 69, wherein 1 to 200 milligrams of the statin is present in the unit dose.

73. (original) The composition according to claim 68 or 69, wherein 5 to 80 milligrams of the FLAP inhibitor is present in the unit dose.

74. (original) A method of reducing C reactive protein (CRP) in a human subject, comprising:

administering to a human in need of treatment to reduce CRP a composition according to any one of claims 55-73 in an amount effective to reduce serum C reactive protein in the human subject.

75. (original) The method of claim 74, comprising:

selecting for the administering step a human subject at risk for a disease or condition selected from the group consisting of myocardial infarction, acute coronary syndrome, stroke, or peripheral arterial occlusive disease.

76. (original) The method of claim 74 or 75, wherein the composition is administered in an amount effective to reduce serum LDL and serum leukotrienes in the human subject.

77. (original) A method of reducing C reactive protein (CRP) in a human subject, comprising:

selecting a human subject that receives statin therapy to reduce serum LDL, wherein the statin therapy optionally reduces serum CRP in the human subject; and

administering to the human subject a leukotriene synthesis antagonist, in an amount effective to further reduce CRP in the human subject.

78. (original) A method of reducing C reactive protein (CRP) in a human subject, comprising:

identifying a human subject in need of treatment to reduce serum CRP;

administering to the human subject a composition comprising a statin;

administering to the human subject a composition comprising a leukotriene synthesis inhibitor,

wherein the statin and the leukotrience synthesis inhibitor are administered in amounts effective to reduce serum CRP in the human subject.

79. (original) A method according to claim 78, wherein the identifying comprises identifying a human subject that exhibits one or more risk factors for myocardial infarction, acute coronary syndrome, stroke, or peripheral arterial occlusive disease.

80. (original) A method according to claim 78, wherein the identifying comprises measuring CRP in the human subject.

81. (original) A method according to any one of claims 78-80, wherein the statin and the leukotriene synthesis inhibitors are simultaneously administered.

82. (original) A method according to any one of claims 78-81, further comprising:

measuring serum C-reactive protein in the human subject to monitor therapeutic efficacy of the administering.

83. (original) A method according to any one of claims 78-82, further comprising modifying the amount or frequency of the administration following the measuring in order to achieve a target measurement of CRP in the human subject.

84. (original) Use of a leukotriene synthesis inhibitor and a statin for the manufacture of a medicament for reducing CRP levels in a human subject.

85. (new) The method of claim 27, wherein, wherein the FLAP polymorphism further comprises marker SG13S25 (SEQ ID NO: 1, position 165553), allele G.

86. (new) The method of claim 85, wherein the FLAP polymorphism further comprises SG13S32 (SEQ ID NO: 1, position 176579), allele A.

87. (new) The method claim 86, wherein the FLAP polymorphism further comprises marker SG13S106 (SEQ ID NO: 1, position 198547), allele G or A.

88. (new) The method claim 30, wherein the haplotype further comprises marker SG13S25 (SEQ ID NO: 1, position 165553), allele G.

89. (new) The method claim 88, wherein the haplotype further comprises SG13S32 (SEQ ID NO: 1, position 176579), allele A.

90. (new) The method of claim 89, wherein the haplotype further comprises marker SG13S106 (SEQ ID NO: 1, position 198547), allele G or A.